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F I N L   R E P O R T

INVESTIGATIONS OF HIP DYSPLASIA
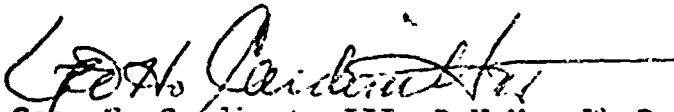IN THE MILITARY WORKING DOG

AFOSR GRANT No. 69-1718

Some illustrations omitted (·

FORWARD

The results of this investigation have established a base for exploring the possible role of abnormal muscle function of the pectineus muscle in the etiology of canine hip dysplasia. This report represents only preliminary results which bear on this question since the length of support which ended in September 1971 was not of sufficient duration to fully evaluate the problem, i.e. the evaluation and correlation of hip dysplasia in adult dogs relative to the status of the pectineus muscle in the neonate. This is a common problem for investigations of canine hip dysplasia due to the late onset of the radiographic disease in many dogs.

This research program is continuing with support from the Morris Animal Foundation and Seeing Eye, Inc. through September 1972. This additional period of support will enable this program to further evaluate the relation of the myopathy to the incidence of hip dysplasia in older dogs. As this information becomes available and the results are published, the AFOSR will be notified and acknowledged for its support.

Geo. H. Cardinet, III, D.V.M., Ph.D.
Principal Investigator
1 March 1972

GRANT PUBLICATIONS

1. Breit, J. F., Cardinet, III, G. H., Tunell, G. L. and Fedde, M. R. Localization of Obturator and Pectineal Motoneurons in the Dog. Am. J. Anatomy, 132:21-32, 1971.

2. Cardinet, III, G. H., Wallace, L. J., Fedde, M. R.. Guffy, M. M., and Bardens, J. W. Normal Growth and Differentiation of Type I Type II Muscle Fibers in the Dog. The Physiologist, 12:192, 1969

3. Cardinet, III, G. H., Wallace, L. J., Fedde, M. R., Guffy, M. M. and Bardens, J. W. Developmental Myopathy in the Canine. Arch. Neurology, 21:620-630, 1969.

4. Cardinet, G. H., Wallace, L. J., Guffy, M. M. and Fedde, M. R. Neuromuscular Disease: A Potential Factor in the Etiology of Canine Hip Dysplasia. AFOSR Publication No. 70-2368TR:89-103, 1970

5. Cardinet, III, G. H., Tunell, G. L. and Fedde, M. R. A Comparative Study of Contractile and Histochemical Properties in Normal and Hypotrophic Muscle in the Dog. Anat. Record, 169:288-289, 1971.

6. Cardinet, III, G. H., Fedde, M. R. and Tunell, G. L, Correlates of Histochemical and Physiological Properties in Normal and Hypotrophic Pectineus Muscles of the Dog. Accepted for publication in Laboratory Investigation, see Appendix I.

7. Tunell, G. L., Cardinet, G. H. and Fedde, M. R. Changes in Postnatal Contraction and Half-Relaxation Times of the Canine Pectineus Muscle. Am. J. Vet. Res. 33:127-130, 1972.

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11. Wallace, L. J., Guffy, M. M. and Cardinet, III, G. H. Pectineus Tendon Surgery for Treating Canine Hip Dysplasia. Accepted for publication in Proceedings of A. A. H. A. 39th Ann. Meeting, 1972, see Appendix II.

STATEMENT OF THE PROBLEM

Hip dysplasia is the number one veterinary medical problem of military working dogs.[1,2] Hip dysplasia is a hereditary disease common to many breeds of dogs of large body size. A notable example is the German Shepherd Breed in which the incidence of the disease may be 50 percent or greater.[3-5] The disease is diagnosed by radiographic examination. The disease cannot be diagnosed at birth and in most cases radiographic diagnosis is not possible until 3 - 4 months of age in severe cases. Studies indicate that at 1 year of age only 69 percent of those dogs which will eventually become dysplastic can be detected by radiographic examination.[6] The accuracy of detection by radiographic examination increases to 95 and 98 percent at 2 and 3 years of age respectively. Therefore, the accuracy of detecting dysplastic-free dogs before 1 year of age is very limited.

The high incidence of the disease and wide range of ages when radiographic examination permits diagnosis presents a serious problem. Today, the armed services cannot fill induction quotas with dysplastic-free dogs.[1,2]

As with any disease problem, control and eradication are the most effective means to a solution. The introduction of therapeutic measures for control and eradication can only be done after the etiology and pathogenesis of a disease are established. Without a fundamental knowledge of the etiology

and pathogenesis, control measures can be expected to be
only partially effective. The etiology and pathogenesis of
canine hip dysplasia are not known and current control
measures are only partially effective. Until the etiology
and pathogenesis of canine hip dysplasia are determined, the
military working dog program can anticipate hip dysplasia to
continue to be its number one veterinary medical problem.

THE RESEARCH PROGRAM

A. Introduction

While many aspects of hip dysplasia have been studied
and described, the etiology and pathogenesis of the disease
have remained undefined. The only common denominator which
has come out of these studies appears to be that joint laxity
precedes and results in the wide variety of acetabular and
femoral changes that are observed.[5] Thus, studies of
factors which contribute to joint laxity offer promise of
determining the etiology of the disease. In 1968, a palpation
technique was developed to judge joint laxity in puppies.[7]
In the development of this technique it was noted that
abduction was restricted in potential hip dysplastic puppies
and it appeared that the pectineus muscle was responsible
for the restricted abduction. Thus, there was a suggestion
of a muscle abnormality possibly being associated with hip
dysplasia. Skeletal muscle changes in canine hip dysplasia

have not received thorough investigation; however, gross
observations suggest an unsatisfactory ratio of pelvic size
to pelvic muscle mass.[8]

B. Objectives

Generally, the objectives of the research were to
investigate the nature of the pectineal myopathy and inquire
into its possible role as an etiological factor in the
pathogenesis of canine hip dysplasia.

More precisely the objectives were:

1. To define the pectineal myopathy as to its incidence
   and extent in German Shepherd dogs, and

2. To determine the etiology of the pectineal myopathy,
   and,

3. To determine the chronological relationships of the
   lesion as to onset and sequelae, and

4. To determine the heritability and mode of inheritance
   of the developmental myopathy, and

5. To determine if diagnostic procedures may be developed
   to detect the myopathy, less than performing pectineal
   biopsies or myectomies, and

6. To correlate the incidence of the pectineal myopathy
   with laxity of the coxofemoral joint by palpation, and

7. To correlate laxity of the coxofemoral joint in
   puppies with the subsequent development of the coxo-
   femoral joint, and

8. To determine the incidence or relationship of the
developmental myopathy with subsequent development
of the hip joint.

C. Results

1. The Developmental Myopathy

The results of this investigation clearly established
that a developmental myopathy of the pectineus muscle was
present in young dogs. The myopathy was characterized by
hypotrophy (lack of growth) of Type II muscle fibers, which
is best demonstrated by the histochemical identification of
muscle fibers using the myofibrillar ATPase reaction. The
earliest age at which the lesion has been observed is 27 days
of age. It has not been possible using the techniques
employed to identify abnormal muscles before this age. The
lesion appears to be transient since it has not been observed
in dogs over 103 days of age.[9,10,11]

To date, 283 dogs have been examined between 27 and 103
days of age. The lesion has been observed in German Shepherds,
German Shepherd-Greyhound crosses, and mongrels but not in
Greyhounds (Table 1). It is of considerable interest that the
lesion has not been found in Greyhounds since this breed is
relatively free of hip dysplasia. While this observation does
not prove that there is a relationship between the incidence
of hip dysplasia and the muscle lesion, it is an observation
that would be consistent with such a relationship. The extent

TABLE 1. Incidence of developmental myopathy (Type II fiber hypotrophy) in the pectineus muscles of dogs, 27 to 103 days of age.

Breed	Number Examined	Number of Normal	Number of Abnormal	Percent Affected
German Shepherd	185	19	166	90.0
Greyhound	22	22	0	0.0
Greyhound-Shepherd	26	13	13	50.0
Mongrel	50	33	17	34.0

TABLE 2. Percent frequencies of myopathy scores in German Shepherd dogs, 27 to 103 days of age.

		Myopathy Score				
	Normal	Minimal	0-25%	25-50%	50-75%	75-100%
Percent	10	15	33	17	11	14

of the myopathy varies between muscles. Therefore, a subjective
scoring system has been devised to classify the lesion. A lesion
was scored 0-25%, 25-50%, 50-75% or 75-100% based on the subjective
assessment of the percent total cross-sectional area of the muscle
that was involved. Another classification was "minimal" in which
the status of the muscle was in doubt; in this instance a few small
type II fibers were observed but overt signs of type II fiber
hypotrophy were not present. This classification may include
normal muscle and represent the normal variation which can be
anticipated. The frequency distributions of these myopathy scores
are presented in Table 2 for German Shepherd dogs, 27-103 days
of age.

Our studies have established that the myopathy results
in altered physiological properties of the pectineus muscle,[12] a
prerequisite for any thesis that abnormal function of this muscle
may influence the development of the coxofemoral joint (refer to
Appendix I). The histochemical and isometric contractile
properties of normal and hypotrophic pectineus muscles were
studied in 21 German Shepherd dogs at 56 to 60 days of age. The
hypotrophic muscles had longer contraction and half-relaxation
times and developed smaller maximum isometric twitch and tetanic
tensions. Correlates between the contractile, histochemical
and morphological properties of the muscles were observed. The
inverse of contraction times (a presumed estimate of speed of
contraction) were directly proportional to the relative type II
fiber composition of the muscles. The maximum isometric twitch
and tetanic tensions developed by the muscles were directly

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proportional to the mean cross-sectional area of the fibers comprising the muscles. Thus, the differences in contractile properties between normal and hypotrophic muscles were directly attributable to the effect of the disease on fiber size.

In summary, our studies have established a pathological and functional basis for investigations into the possible relationship between abnormal muscle function and the development of hip dysplasia, and constitutes the working hypothesis of this research program (Figure 1).

2. Etiology of Pectineal Myopathy

We have been unable to definately establish the etiology of the pectineal myopathy. The preferential effect of the disease on type II fibers coupled with the knowledge that motor units are homogenous[13,14] with respect to fiber types, suggests that the disease is neural in origin. However, pathological studies of peripheral nerves and lumbar spinal cords have not revealed overt signs of neural pathology.

The canine pectineus muscle is described as being innervated by the obturator nerve.[15] In order to precisley evaluate the lumbar spinal cord neurons, a study was conducted to localize the obturator and pectineal motoneuron columns.[16] The obturator motoneuron column was located in segments L4 through L6 while the pectineal column was located in segments L4 and L5 only. With this knowledge, lumbar spinal cords were examined in segments L4 through L6 and overt pathological changes were not observed. A study of neuronal phosphorylase activity revealed variations in phosphorylase activity of alpha motoneurons in the dog. No
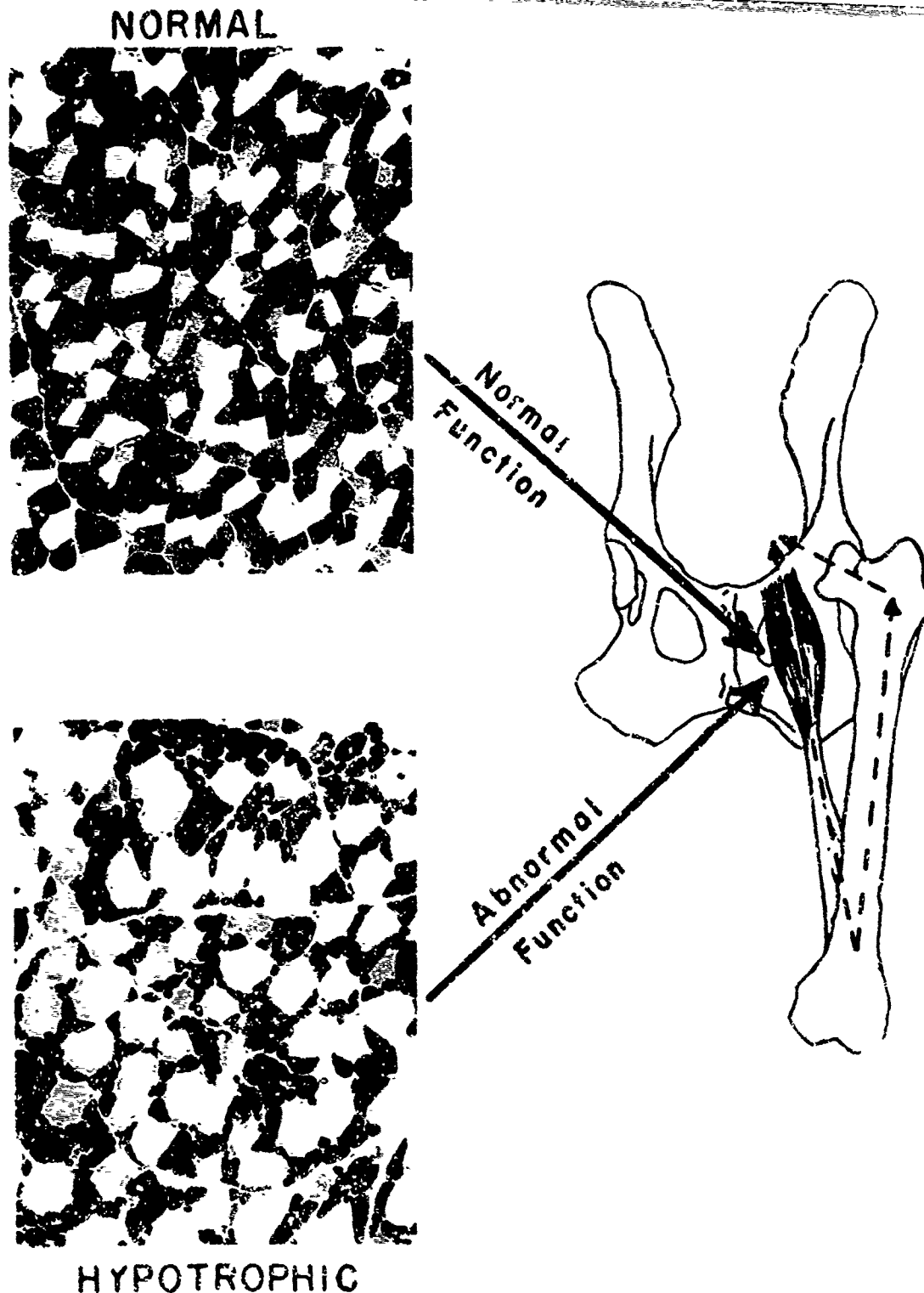
NORMAL

HYPOTROPHIC

Figure 1. Hypothesis of muscle dysfunction as an etiological
factor in the pathogenesis of canine hip dysplasia. Normal
muscle (upper left) exerts tensions compatible with the normal
development of the coxofemoral joint. Hypotrophic muscle (lower
left) exerts tensions incompatible with the normal development
of the coxofemoral joint. Vectors of tensions developed by the
pectineus muscle are indicated (---). Sections stained for
myofibrillar ATPase in which Type I fibers are light staining
and Type II fibers are dark staining. The hypotrophic muscles
are characterized by hypotrophy of Type II fibers and hypertrophy
of some Type I fibers.

such variation has been observed in the cat.[17] However, the variation in alpha motoneuron phosphorylase activity was not restricted to the obturator or pectineal motoneuron columns. Therefore, the significance of this observation is obscure.

Recently we have observed that the pectineus muscle receives a variable motor innervation from the femoral nerve, in addition to the obturator nerve. To date, 10 of 32 German Shepherd dogs have been observed to have dual innervation of the pectineus muscle. The significance of this observation is unknown but is currently being analysed along with data on the myopathy.

3. Chronology of Lesion, Onset and Sequelae

While the incidence of the developmental myopathy is approximately 90% in German Shepherd dogs,27-103 days of age, pathology of the pectineus muscle is infrequently seen in German Shepherd dogs over 100 days of age. Therefore the factor or factors responsible for the condition appear to be corrected between 8 weeks of age and sometime around 14-16 weeks of age. A study has been initiated to determine the chronological changes during this period of growth and differentiation of the pectineus muscle. A few cases have revealed pathology of the pectineus muscle at 112 and 168 days of age. The lesion observed has been characterized by type I fiber grouping which is suggestive of collateral reinnervation of the muscle. This finding would be consistent with the presupposed origin of the myopathy being neural in nature. However, in most instances pathology of the muscle is not observed after 16 weeks of age. Therefore, any

relationship between the myopathy and the development of hip
dysplasia may be influenced by sequelae of the lesion after
8 weeks of age. This study is still in progress and the results
are therefore incomplete.

4. Heritability of Myopathy

Some insight into the heritability of the myopathy is
possible. In an analysis of 138 pups (32 litters), 68 females
and 70 males, the severity of the myopathy was greater in males
than females (Table 3). On a 6 point scale of severity (normal =
1; minimal = 2; 0-25% =3; 25-50% = 4; 50-75% = 5; and 75-100% = 6),
male pups averaged 3.73 whereas females averaged 2.99.

Sixteen of the 32 litters were from parents of known pheno-
types with respect to the myopathy. These are summarized in
Table 4. The realized heritability in these data is about 0.12.
The severity of the myopathy was greater in progeny of two abnormal
parents than in progeny of normal parents.

Analysis of variance of myopathy scores for 74 pups from
random parents yielded the following estimates of variance
components (Table 5). Heritability estimated from half-sib
correlation is 0.24. Commonality of enviornment or dominance
deviations increased the similarity among maternal sibs. Thus,
this analysis suggests a moderate effect of heredity in causing
differences in myopathy scores.

In summary, the data at hand are adequate to establish that
males are affected more severely than females. The data also
show that though the myopathy is not transmitted as a simple
dominant or recessive trait, it is moderately influenced by

TABLE 3. Sex differences in severity of myopathy in German
Shepherd pups at 8 weeks of age (32 litters)

Sex	No.	Frequency percentages of myopathy grades					
		Normal	Minimal	0-25%	25-50%	50-75%	75-100%
Male	70	3	16	34	17	13	17
Female	68	13	24	37	13	4	9

TABLE 4. Progenies from parents of known phenotype with respect
to the myopathy grade (Sixteen Litters)

Mating Type	Sex of Progeny	Normal	Minimal	0-25%	25-50%	50-75%	75-100%
Abnormal ♂ x	male	0	2	10	2	2	5
Abnormal ♀	female	2	3	7	2	1	2
(8 Litters)	TOTALS.......	2	5	17	4	3	7
Abnormal ♂ x	male	1	0	2	0	1	0
Normal ♀	female	2	2	1	0	0	0
(3 Litters)	TOTALS.......	3	2	3	0	1	0
Normal ♂ x	male	0	0	0	0	0	0
Abnormal ♀	female	1	1	1	1	0	0
(1 Litter)	TOTALS.......	1	1	1	1	0	0
Normal ♂ x	male	0	1	5	2	1	1
Normal ♀	female	0	4	1	1	0	0
(4 Litters)	TOTALS.......	0	5	6	3	1	1

TABLE 5. Myopathy variance components and their genetic interpretation

Component	Source	Value	Genetic Interpretation
σ^2_e	Residual-primarily among pups within litters	1.59	$\frac{1}{2} V_a + 3/4 V_D + V_{ew}$
$\sigma^2_{L:S}$	Among litters within sire groups	0.53	$\frac{1}{2} V_a + \frac{1}{4} V_D + V_{ec}$
σ^2_S	Among sire progeny groups	0.15	$\frac{1}{4} V_a$
σ^2_M	Among sexes	0.23	

hereditary differences. Heritability of the trait is estimated provisionally at between about 11 to 25 per cent. Strong maternal effects of some sort also were present. However, the standard errors of these estimates are quite large and thus many more data are needed before relaiable estimates can be obtained.

5. Diagnostic Procedures for Detection of Myopathy

We have studied the possibility of detecting pups with the pectineal myopathy by means other than biopsy or myectomy and the use of histochemical evaluation.

Serum enzyme activities such as creatine phosphokinase and glutamic-oxaloacetic transaminase are frequently elevated in diseases of muscle.[18] We have not observed significant differences in creatine phosphokinase or glutamic-oxaloacetic transaminase activities between normal and hypotrophic muscled pups.

Electromyography is frequently a useful adjunct to the diagnosis of neuromuscular diseases. Preliminary studies of electromyographic characteristics in normal and hypotrophic muscles have not revealed differences. Fibrillation potentials which are characteristic of denervation have not been observed and motor unit recruitment following stretching appears to be similar in normal and hypotrophic muscles. Therefore, diagnostic procedures for the detection of the myopathy short of histochemical analyses have not been developed.

6. Incidence of Myopathy and Joint Laxity

Laxity of the coxofemoral joints has been estimated in 84 German Shepherd puppies in which the pups were palpated by 3 of the investigators. The palpation scores listed represent the summed estimates of millimeter displacement of the femoral head from the acetabulum for right and left joints by all three investigators. The results of these estimates are presented in Table 6. Comparisons of laxity determined for right and left coxofemoral joints indicate no significant difference between sides (Table 7). A comparison of laxity observed and the myopathy grade indicate no significant difference is observed. Therefore, joint laxity does not appear to be related to severity of the myopathy (Table 8).

7. Relationship of Joint Laxity with Hip Dysplasia

The age of the dogs studied to date limits the analysis of joint laxity with respect to hip dysplasia. However, it is possible to test for differences in the determination of joint laxity at 8 weeks of age and the development of hip dysplasia at 6 months of age. Based on radiographic diagnoses at 6 months of age, significant differences were noted between radiographic normal, suspect and dysplastic dogs and their palpation scores (Table 9). Though small, dogs with normal hips at 6 months of age had smaller estimates of joint laxity than suspect or dysplastic dogs.

8. Relationship between Myopathy and Incidence of Hip Dysplasia

Our analysis of lesion incidence and the development of hip dysplasia is limited to radiographic diagnosis at 6 months. As the colony ages, the observations can be extended to radiographic

TABLE 6. Palpation scores of 84 German Shepherd dogs.

	Palpation Score (mm. displacement)[*]
Mean	11.8
Range	4.0 - 20.5
S.D.	3.60
S.D.$_p$	3.62
S.E.$_m$	0.39

* Summation of displacement estimated by three investigators for both right and left coxofemoral joints.

TABLE 7. Comparisons of palpation scores of right and left hips in 84 German Shepherd dogs.

	Palpation Score (mm. displacement)[*]	
	Right Hip	Left Hip
Mean	5.7	6.2
Range	1.0 - 10.0	2.0 - 10.5
S.D.	1.88	1.94
S.D.$_p$	1.90	1.96
S.E.$_m$	0.21	0.21

Comparison	t-value	P
Right : Left	1.6226	> 0.05

* Summation of displacement estimated by three investigators for one coxofemoral joint.

TABLE 8. Comparisons of palpation scores and myopathy grades

		MYOPATHY GRADE					
		Normal n=9	Minimal n=13	0-25% n=31	25-50% n=11	50-75% n=7	75-100% n=13
Palpation Score	Mean	13.2	9.6	11.8	13.4	10.3	12.5
	S.D.	2.90	3.10	3.12	4.54	3.43	3.36
	S.D.$_p$	3.08	3.22	3.17	4.77	3.71	3.49
	S.E.	1.03	0.89	0.57	1.44	1.40	0.97

Comparisons	t-value	P
Normal : Minimal	2.5793	< 0.02
Normal : 0-25%	1.1166	> 0.05
Normal : 25-50%	0.1371	> 0.05
Normal : 50-75%	1.6583	> 0.05
Normal : 75-100%	0.4719	> 0.05

TABLE 9. Comparisons of palpation scores and 6 month radiographic diagnosis

		6 Month Radiographic Diagnosis		
		Normal n=27	Suspect n=18	Dysplastic n=14
Palpation Score	Mean	10.1	12.2	13.5
	S.D.	2.97	2.90	3.24
	S.D.$_p$	3.03	2.99	3.36
	S.E.$_m$	0.58	0.70	0.90

Comparisons	t-value	P
Normal : Dysplastic	3.1296	< 0.01
Normal : Suspect	2.3110	< 0.05
Suspect : Dysplastic	1.0875	> 0.05

diagnoses at older ages. To date our results do not provide information on these parameters.

D. Related Research Efforts

1. Surgical Rehabilitation of the Dysplastic Dog

While not stated as an objective in our original research proposals, a new surgical procedure has been developed which has proven to be very effective in rehabilitating clinically dysplastic dogs.[19,20,21] Also, refer to Appendix II. The new procedure developed consists of performing tenotomy or tenectomy of the pectineus muscle in dysplastic dogs. This operation has resulted in dramatic improvement of the functional use of the legs without evidence of pain in approximately 90 per cent of the cases. This surgical procedure offers immediate application of research findings to the welfare of the Military Working Dog.

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APPENDIX   I

CORRELATES OF HISTOCHEMICAL AND PHYSIOLOGICAL PROPERTIES IN

NORMAL AND HYPOTROPHIC PECTINEUS MUSCLES OF THE DOG

by

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INTRODUCTION

Histochemical studies of skeletal muscle fibers have revealed striking enzymic differences among muscle fibers which have resulted in numerous classifications of fiber types. [17,21,22,30,34] Physiological studies have demonstrated slow, fast, and intermediate contracting motor units, [14,15,26,36] each of which appears to be composed of a uniform fiber type. [9,20,36] These observations imply that a muscle containing more than a single fiber type is composed of fibers which are biochemically and functionally heterogeneous.

Histochemical classifications of fiber types have been based on different enzymic procedures in a variety of muscles and species. It is therefore often difficult to equate muscle fiber types defined by the different classification criteria and make correlations with functional data. [7,37] The histochemical method which offers the greatest promise for correlative studies of fiber types with respect to contraction times of muscles, is the myofibrillar adenosine triphosphatase (ATPase) method of Padykula and Herman [27,28] since quantitative measurements of myosin and myofibrillar ATPase activities are inversely proportional to contraction time; i.e. faster muscles have higher myosin and myofibrillar ATPase activities. [2,3,4,8,33] Further, the intrinsic speed of shortening of sarcomeres and the actin-activated ATPase activity of myosin are directly proportional. [4] When the myofibrillar ATPase histochemical method is applied to skeletal muscle and fibers are classified according to the nomenclature of Engel, [21,22] two

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principal fiber types (referred to a   and type II fibers)

are differentiated. Type I fibers ar.   . staining (low

reacting) and type II fibers are dark s   ..ing (high reacting);

fibers with intermediate staining may also be observed.

Recently in a study of individual motor units of the cat, fast

contracting units were demonstrated to be uniformly composed

of dark staining fibers as judged by myofibrillar ATPase

histochemistry.[9]

The myofibrillar ATPase method is preferred for typing

fibers in the study of many pathological conditions since fibers

classified by their myofibrillar ATPase reaction retain their

classification characteristics while reactions for phosphorylase

and mitochondrial enzymes may be altered.[22] During the neonatal

development of pectineal muscles in some young dogs, there is a

retardation in the growth and differentiation of some muscle

fibers, particularly type II fibers.[12] This type II fiber

hypotrophy is being investigated in this laboratory to explore the

possible role of abnormal muscle function as an etiological

factor in the pathogenesis of canine hip dysplasia, (a developmental

abnormality of the coxofemoral joint). The purpose of the present

study was twofold: 1) to determine if the type II fiber hypotrophy

altered the physiological properties of the pectineus muscle, a

prerequisite for any thesis that abnormal function of this muscle

may influence the development of the coxofemoral joint, and 2)

to correlate the physiological observations with the morphological

and histochemical properties of the muscles since the type II

fiber hypotrophy results in different relative contributions of

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type I and type II fibers with respect to the total composition of the muscle. Evidence will be presented to show that the hypotrophy results in changes in both the time course and magnitude of isometric contractions and that these changes may be directly related to the type and size of fibers comprising the muscles.

METHODS

General outline

Twenty-one (11 male, 10 female) German Shepherd dogs, 56 to 60 days old were studied. At this age postnatal changes in contraction and relaxation times of the pectineus muscle have reached a plateau.[35] The dogs were weighed and anesthesia was induced by intravenous administration of 5% sodium pentobarbital (29 mg/kg). Additional anesthetic was administered, as needed, to maintain a surgical plane of anesthesia during the recording of isometric contractions. At the conclusion of the recordings, the muscles were removed, weighed, and processed for histochemical analyses. Statistical analyses of the data included the use of the Student's t-test, regression analysis and correlation analysis.[1]

Physiological procedures

The dogs were placed in dorsal recumbency. The femurs were fixed to the table, perpendicular to the longitudinal axis of the body, by metal pins inserted in their distal ends. This prevented movement of the pelvis so that isometric contractions could be recorded. The pectineal muscles were isolated through a skin incision and dissected free except for tendons

of origin. The pectineal branches of the obturator nerve were transected; the blood supply left intact. The margins of the skin incision were sutured to a metal ring to form a pocket into which mineral oil was poured to prevent dehydration. The mineral oil was maintained at $38 \pm 1.5^{\circ}C$. The tendon of insertion was connected to a strain gauge transducer (Grass FT03C) with tungsten wire (125 u diameter). The transducer had sufficient dynamic frequency response to follow a sinusoidal oscillation of 500 Hz produced by an electromagnetic vibrator (M. B. Electronics Model PM-50). The strain gauge was mounted on a manipulator which permitted alignment and passive tension adjustment. Isometric tensions generated by the muscle were detected by the transducer and recorded on a direct-writing recorder (Sanborn Model 150). The recorder had sufficient frequency response to faithfully reproduce the fastest twitch as determined by identical tracings from an oscilloscope (Tektronix Model 564) connected in parallel to the recorder. Electrical stimulation (square-wave pulses of 0.1 msec. duration) was applied to the muscles by two stainless steel electrodes placed on opposite sides of the muscle belly. The muscles were stimulated directly since the canine pectineus muscle, in addition to receiving motor innervation from the obturator nerve, receives variable motor innervation from the femoral nerve.[11] The femoral branch, when present, was transected in the process of dissecting the muscle free for the recording sequence. The optimal stimulus voltage was determined for each muscle by applying single, graded stimuli until higher voltages did not increase the twitch tension; the lowest voltage that yielded a maximal twitch tension (range 8 to 29 volts) was then used and monitored on an oscilloscope

throughout the experiment. The optimal passive tension applied
to the muscle during the experiment was determined for each
muscle. The muscle was stimulated at graded, increasing passive
tensions until a maximum twitch tension was developed using the
optimal voltage; the lowest passive tension which produced a
maximum twitch tension was used throughout the experiment. After
the optimum stimulus and passive tension were determined, the
muscle was stimulated and a simple twitch recorded, from which
these parameters were measured: maximum isometric twitch tension,
contraction time, and half-relaxation time. Next, the response
to repetitive stimuli was determined by stimulating the muscle
at frequencies of 5 to 100 Hz for 1 second durations from which
the maximum isometric tetanic tension was determined and
tetanus:twitch ratios were calculated. The muscle was then
stimulated and a simple twitch recorded, from which the maximum
isometric twitch tension and contraction time were measured
for comparison with the isometric twitch prior to tetanus.

Morphological and histochemical procedures

Immediately after conclusion of the recording sequence
the muscles were removed, weighed and held at $4^{\circ}C$ for 30 minutes.
Transverse sections were cut through the entire muscle belly
with a razor blade and frozen in 2-methylbutane cooled to
$-125^{\circ}C$ to $-150^{\circ}C$ in liquid nitrogen. Cryostat sections,
10 u thick, were cut and incubated for the demonstration of
myofibrillar ATPase according to the method of Padykula and
Herman[26,27] as previously described.[19] This method applied

to the canine pectineus muscle differentiates 3 fiber types: type I fibers (light staining), type II fibers (dark staining) and intermediate fibers (intermediate staining between type I and type II fibers). Sections were examined by light microscopy and the muscles classified as normal or hypotrophic. Since the extent of involvement can vary from focal to diffuse,[12] the hypotrophic muscles were further classified subjectively. The lesions were classified as focal if the extent of involvement was judged to be less than 50 percent of the cross-section of the muscle belly, and diffuse if judged to be greater than 50 percent.

Sections from the mid-belly of all right muscles were photographed and cross-sectional area of muscle fibers measured with a planimeter; approximately 1000 fibers (880-1362) were measured from each muscle, a total of 22,812 fibers being measured. Based on these measurements the mean fiber area of each fiber type, the percentage of each fiber type and the relative percent area of each fiber type comprising the muscles were determined.

Abbreviations and definitions

P_t Maximum isometric twitch tension.

P_o Maximum isometric tetanic tension.

P_o/P_t Ratio of maximum isometric tetanic tension to maximum isometric twitch tension.

T_c Isometric twitch contraction time, i.e. the time elapsed from onset to the peak of an isometric twitch.

$T_{\frac{1}{2}r}$ Isometric twitch half-relaxation time, i.e. the time elapsed from peak to one-half peak tension of an isometric twitch.

i Subscript denoting that value is derived for type I, type II or intermediate fibers, where i may be I, II or int respectively.

$\%_i$ Percentage of each fiber type.

A_i Mean cross-sectional area of each fiber type.

$\%A_i$ Percent area of each fiber type comprising the muscle (derived from the product of the percentage of a given fiber type and its mean fiber area divided by the summed products of all three fiber types, where

$$\%A_i = \%_i \times A_i \div \left[(\%_I \times A_I) + (\%_{int} \times A_{int}) + (\%_{II} \times A_{II}) \right] .$$

RESULTS

Morphological and histochemical observations

Fourteen of the 21 dogs were abnormal, their pectineus muscles having the characteristic lesion of type II fiber hypotrophy (Fig. 1, A & B). Seven were judged to have focal lesions while seven were diffuse. The body weights of the normal and hypotrophic groups of dogs were not significantly different; however, the absolute and relative muscle weights of the hypotrophic muscles were smaller (Table 1).

(Fig. 1A, B and Table 1 near here)

Percentages of each fiber type were similar for both normal and hypotrophic muscles; however, mean fiber cross-sectional areas differed. All fiber types in hypotrophic muscles were

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smaller in mean cross-sectional area (Table 2). Megahistograms, the summed individual frequency distributions of type I and type II fibers for each group,[6] illustrate the preferential effect of the disease on the cross-sectional area of type II fibers (Figs. 2, 3). Type I fibers are affected, which may be noted by a shift to the left (Fig. 2); however, this shift is greatest for type II fibers (Fig. 3). A small percentage of type I fibers were hypertrophic (Fig. 2); the largest type I fibers measured in the normal muscles were 1350 u^2 while in the hypotrophic muscles some type I fibers were 1800 u^2. Since the disease has a preferential effect on the cross-sectional area of type II fibers, the relative contribution of each fiber type to the total muscle varies. This resulted in the percent area of type I and type II fibers to be significantly different in the normal and hypotrophic muscles in that hypotrophic muscles had larger percent areas for type I fibers or, conversely, smaller percent areas for type II fibers.

(Figs. 2, 3 and Table 2 near here)

Physiological observations

The means and standard errors of the optimal stimulus voltages were 16.1 $\pm$ 1.1 and 17.0 $\pm$ 0.7 for the normal and hypotrophic muscles, respectively; these means were not significantly different. Significant differences were observed between the isometric contraction characteristics of the normal and hypotrophic muscles with the exception of P_o/P_t ratios which were not significantly different. While there was considerable variation in the values observed for the hypotrophic

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muscles, they tended to have longer contraction and relaxation times, and developed smaller twitch and tetanic isometric tensions (Table 3). The stimulus frequencies required to produce maximum isometric tetanic tensions differed between groups. The mean frequencies and standard errors were 51 ± 2 and 42 ± 2 for the normal and hypotrophic muscles, respectively (P < 0.001). The stimulus frequencies required to produce maximum isometric tetanic tensions were inversely proportional to the contraction times of the muscles; the correlation coefficient was 0.81 (P < 0.001). Thus, faster contracting muscles required higher stimulus frequencies to develop maximum isometric tetanic tensions. There was no significant difference in the pretetanic and posttetanic isometric twitch tensions and contraction times.

(Table 3 near here)

Correlates of morphological, histochemical and physiological observations

The parameters of T_c, P_t, and P_o were related to the histochemical and morphological properties of the muscles. The inverse of contraction time ($1/T_c$) was proportional to $\%A_{II}$ (Fig. 4); the correlation coefficient was 0.97 (P < 0.001). There was no significant correlation between T_c and P_o/P_t ratios.

The maximum isometric twitch and tetanic tensions were proportional to the mean fiber cross-sectional area of the muscles (Figs. 5, 6); the correlation coefficients were 0.89 (P < 0.001) and 0.91 (P < 0.001) respectively. There was no significant

difference between the maximum isometric twitch or tetanic tensions developed and the proportion of type I and type II fibers comprising the muscles.

(Figs. 4, 5 and 6 near here)

DISCUSSION

This study establishes that hypertrophy of the pectineus muscle results in quantitative changes in the time course and magnitude of isometric contractions. Further, these changes may be directly correlated with and probably related to the manifestation of the disease process on skeletal muscle fiber size: 1) with greater retardation in the growth of the fibers there was a corresponding decrease in maximum isometric contraction capability and 2) with the preferential retardation of type II fiber growth there was a resultant change in the relative type I and type II fiber composition of the muscles which was correlated with differences in contraction times.

The intrinsic speed of sarcomere shortening is proportional to the inverse of contraction time.[13,14,16] The relationship between the $\%A_{II}$ of a muscle and the inverse of contraction time observed in our study is likely due to differences in the intrinsic speed of shortening of the muscle fibers. Consistent with this suggestion, the frequencies required to produce maximum isometric tetanic tensions were inversely proportional to the contraction times. However, our data does not present evidence that the intrinsic speed of sarcomeres or the force:velocity properties of either type I or type II fibers are different. The different contraction times were probably not associated with changes in the duration of the active state or series compliance since proportional changes in P_o/P_t ratios

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were not observed. Proportional changes in P_o/P_t ratios would be anticipated with changes in these parameters.

A relationship between the myofibrillar ATPase fiber types and contraction time has been observed in the guinea pig.[29] Barnard et al.[5] studying the contractile and histochemical properties of guinea pig muscle chose to correlate the contractile properties with fiber type classifications based on NADH-diaphorase histochemistry rather than the myofibrillar ATPase reaction; however, their data may also be analyzed in a similar manner as our study. It was possible to approximate the $\%A_{II}$ of the soleus, medial gastrocnemius, flexor digitorum longus, flexor hallucis longus and red portion of vastus lateralis muscles from photomicrographs presented[5] and test for a correlation between $\%A_{II}$ and the inverse of the mean contraction times. The $\%A_{II}$ of these muscles we determined to be 0, 71, 80, 94 and 98 respectively and the inverse of the mean contraction times were respectively 0.012, 0.045, 0.046, 0.047 and 0.053. From the data the correlation coefficient was determined to be 0.99 ($P < 0.001$).

Quantitative studies of "fast" (extensor digitorum longus) and "slow" (soleus) muscles in the rat have demonstrated a direct proportionality between the speed of contraction and actin-activated myosin ATPase activities,[4] where the ATPase activity and speed of contraction are some 2.3 times greater in the extensor digitorum longus than in the soleus. The results of our study show a similar difference (refer to slope of Fig. 4) in that theoretically a pure type II muscle (i.e., $100\%A_{II}$) would be some 2.8 times faster than a pure type I muscle (i.e., $0\%A_{II}$).

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It has been suggested that the histochemical, myofibrillar ATPase method is not a quantitative estimate of speed of contraction but only a gross indicator.[18,29] However gross an indicator it may be, it does provide quantitative estimates and embraces functional and biochemical data. We suggest that the myofibrillar ATPase method is the method of choice for classifying muscle fibers with respect to speed of contraction. Other histochemical methods utilizing mitochondrial enzymes or phosphorylase may sometimes be related or provide further subclassifications of fiber types, (e.g. fatigue.[9,20]). However, these methods are related to enzyme systems which generate ATP for contraction, while the rate of utilization of ATP during contraction appears to be the specific event related to speed of contraction, which the histochemical, myofibrillar ATPase reaction presumably reflects. Further, the myofibrillar ATPase method for fiber typing is preferred for its clarity of distinguishing fiber types and least apparent change in pathological muscle.[22]

The etiology of the hypotrophy of the pectineus muscle is unknown. It has been suggested that it may be a neuropathic disease of muscle, but not in the usual histologic sense, since pathological changes have not been observed in intramuscular nerve branches and/or lumbar spinal cords and esterase activity with motor end-plate localizations appear similar for normal and hypotrophic fibers.[12] In the original description of the disease it appeared that the hypotrophic muscles contained a higher percentage of type II fibers.[12] This suggested a failure

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or delay in the postnatal type II to type I fiber transformation in addition to the retardation in fiber growth. In the present study we did not observe a significant difference in fiber type percentages between normal and hypotrophic muscles. Therefore, it is not clear if the disease affects postnatal type II to type I transformation. However, our results do indicate that the disease may be more precisely defined as a hypotrophy of all fiber types, the extent of which is greatest in the type II fibers. If the disease is neuropathic and does not affect differentiation, then the neural trophic influences on fiber growth must be distinct from neural trophic influences on differentiation which regulate muscle fiber types as suggested by cross-innervation and neonatal neurectomy experiments.[23,24,25,31,32]

While contraction times and tensions were significantly different between normal and hypotrophic muscles, some hypotrophic muscles were similar to normal with respect to these parameters (Figs. 4-6). Therefore, some affected muscles, primarily those with focal lesions, may not differ greatly from normal. On the other hand, those muscles which we subjectively rated as diffusely affected tended to be considerably different. The smaller tensions produced by the hypotrophic muscles were also smaller per gram of muscle. This could be due to differences in mean fiber length, proportion of connective tissue or intrinsic strength of the contractile material.

In many cases of pectineal hypotrophy, abduction of the hip joint is restricted by an apparent increased tone of the pectineus muscle. This may be analogous to the reduction in muscle power but

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increase in tone observed in upper motor neuron lesions and parkinsonism where there is an atrophy of type II fibers.[19] This is believed to result from the disuse of high threshold phasic motor units (type II fiber units) and increased usage of low threshold tonic motor units (type I fiber units).[19] The pattern of preferential type II fiber hypotrophy and hyrertrophy of some type I fibers in the pectineus muscle would be consistent with such a mechanism for decreased power and increased tone.

If it is valid to relate our observations to the function of the muscle in situ, one would predict that the disease results in a weaker and slower muscle. Therefore, the hypotrophy of the pectineus muscle may potentially influence the development of the coxofemoral joint through diminished tension relationships between the muscle and the joint. The effect of diminished tension relationships on the development of the coxofemoral joint remains to be determined.

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ACKNOWLEDGMENTS

TABLE 1. Body weights, pectineus muscle weights and relative pectineus muscle weights of German Shepherd dogs, 56 to 60 days of age.[a]

	Normal Dogs (no. = 7)	Hypotrophic Dogs (no. = 14)	P
Body wt., (kg)	5.12 ± 1.24	4.15 ± 0.87	>0.05
Pectineus muscle wt., (g)			
right	1.71 ± 0.62	1.11 ± 0.28	<0.05
left	1.70 ± 0.53	1.11 ± 0.26	<0.05
right and left	1.70 ± 0.58	1.11 ± 0.27	<0.01
Relative pectineus muscle			
wt., (g muscle/kg body wt.)			
right	0.33 ± 0.05	0.27 ± 0.06	<0.05
left	0.32 ± 0.03	0.27 ± 0.04	<0.01
right and left	0.32 ± 0.04	0.27 ± 0.05	<0.001

[a]
Mean ± standard deviation. Probability values(P) for differences between means have been determined from t values.

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TABLE 2. Percentages, mean areas and percent areas of muscle fiber types in pectineus muscles of German Shepherd dogs, 56 to 60 days of age.[a]

	Normal Dogs (no. = 7)	Hypotrophic Dogs (no. = 14)	P
Fiber type percentages ($\%_i$)			
$\%_I$	38.4 ± 6.2	41.9 ± 9.6	>0.05
$\%_{int}$	1.5 ± 1.3	2.4 ± 1.5	>0.05
$\%_{II}$	60 l ± 6.7	55.7 ± 9.4	>0.05
Mean fiber area (A_i), u^2			
A_I	499 ± 96	395 ± 98	<0.05
A_{int}	356 ± 76	235 ± 104	<0.01
A_{II}	430 ± 108	228 ± 114	<0.01
All fibers	453 ± 102	298 ± 105	<0.01
Percent areas ($\%A_i$)			
$\%A_I$	42.2 ± 6.4	57.8 ± 12.0	<0.01
$\%A_{int}$	1.3 ± 1.1	1.8 ± 1.0	>0.05
$\%A_{II}$	56.5 ± 7.1	40.4 ± 12.1	<0.01

[a] Mean ± standard deviation. Probability values (P) for differences between means have been determined from t values.

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TABLE 3. Isometric contraction measurements of pectineus muscles from German Shepherd dogs, 56 to 60 days of age.[a]

	Normal Dogs (no. = 7)	Hypotrophic Dogs (no. = 14)	P
T_c, (msec)			
right	41.4 ± 3.8	48.6 ± 10.9	<0.05
left	41.2 ± 3.1	50.0 ± 9.6	<0.01
right and left	41.3 ± 3.5	49.3 ± 10.3	<0.001
$T_{\frac{1}{2}r}$, (msec)			
right	46.8 ± 9.0	61.2 ± 22.8	>0.05
left	42.8 ± 5.6	56.9 ± 23.0	>0.05
right and left	44.8 ± 7.7	59.0 ± 23.0	<0.01
P_t, (g)			
right	370 ± 120	183 ± 120	<0.01
left	352 ± 98	188 ± 115	<0.01
right and left	361 ± 110	185 ± 118	<0.001
P_o, (g)			
right	1366 ± 427	741 ± 418	<0.01
left	1373 ± 439	762 ± 428	<0.05
right and left	1369 ± 433	751 ± 423	<0.001
P_o/P_t ratios			
right	3.75 ± 0.51	4.33 ± 1.00	>0.05
left	3.95 ± 0.78	4.30 ± 0.96	>0.05
right and left	3.85 ± 0.66	4.32 ± 0.98	>0.05
Relative P_t, (g/g muscle)			
right	221 ± 39	154 ± 80	<0.05
left	212 ± 46	159 ± 82	>0.05
right and left	217 ± 43	156 ± 81	<0.01
Relative P_o, (g/g muscle)			
right	819 ± 137	630 ± 270	>0.05
left	809 ± 136	648 ± 296	>0.05
right and left	814 ± 136	639 ± 284	<0.05

[a]Mean ± standard deviation. Probability values (P) for differences between means have been determined from t values.

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Illustration omitted

Fig. 1. Transverse sections of normal (A) and hypotrophic (B)
canine pectineus muscles incubated and stained for the
histochemical demonstration of myofibrillar ATPase. Type I
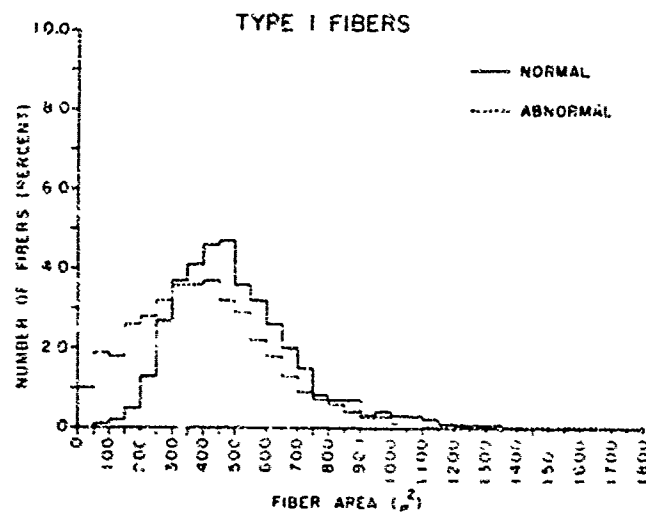fibers are light staining and type II fibers are dark.

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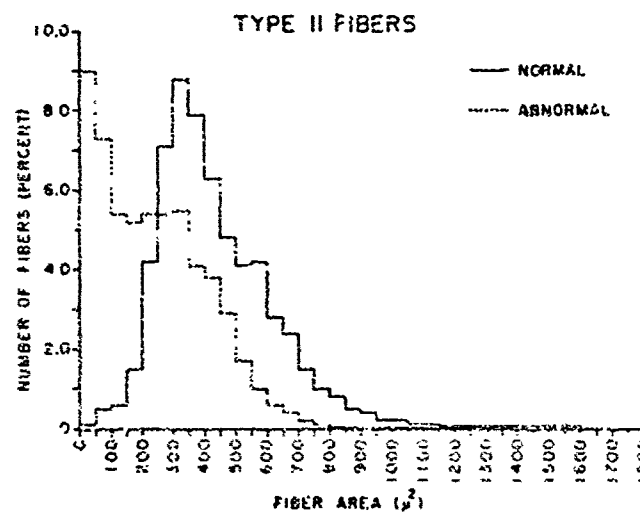Fig. 2. Megahistograms of type I fiber cross-sectional areas in normal and hypotrophic muscles.

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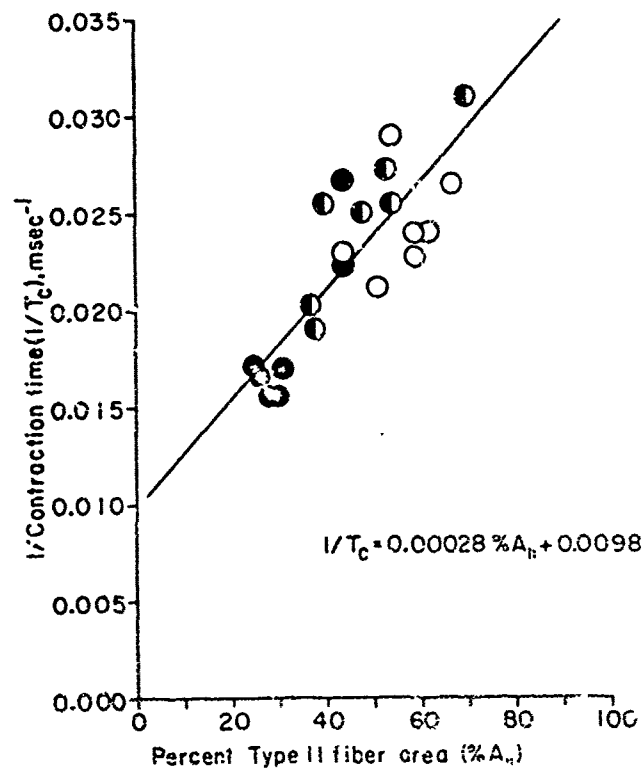**Fig. 3.** Megahistograms of type II fiber cross-sectional areas in normal and hypotrophic muscles.

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Fig. 4. The relationship between the inverse of contraction
time and the percent area of type II fibers in normal and
hypotrophic muscles: O normal muscles, ◑ focally hypotrophic
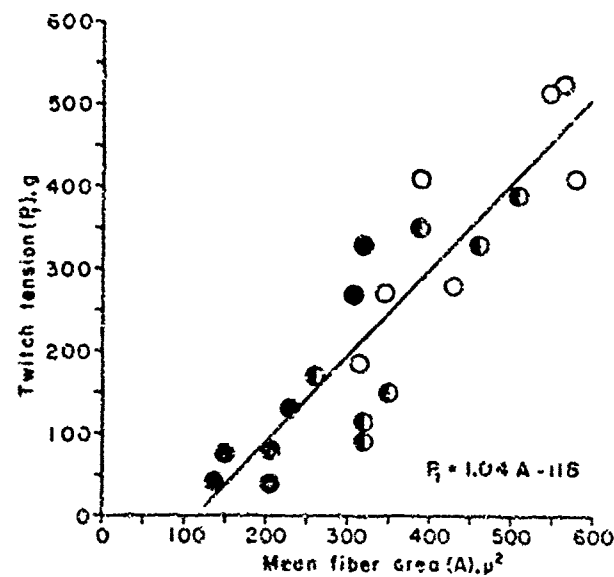muscles, ● diffusely hypotrophic muscles.

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Fig. 5. The relationship between maximum isometric twitch tension and the mean fiber size of normal and hypotrophic muscles: O normal muscles, ◑ focally hypotrophic muscles, ● diffusely hypotrophic muscles.
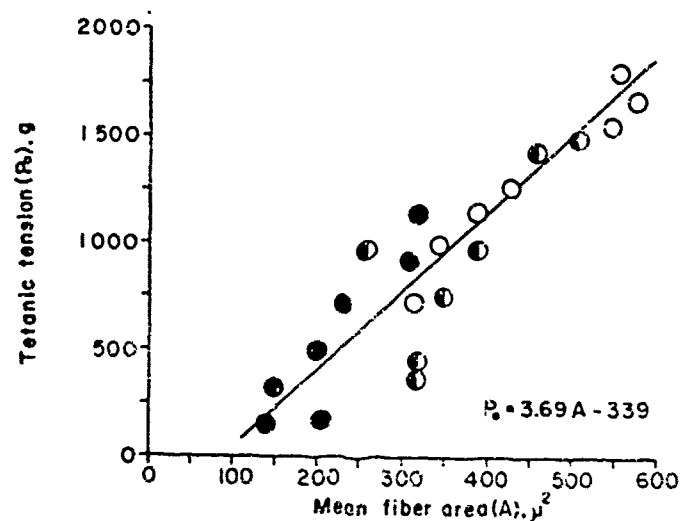
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Fig. 6. The relationship between maximum isometric tetanic tension and the mean fiber size of normal and hypotrophic muscles: O normal muscle, ◐ focally hypotrophic muscles, ● diffusely hypotrophic muscles.

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PECTINEUS TENDON SURGERY FOR TREATING
CANINE HIP DYSPLASIA

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A. Introduction

Since canine hip dysplasia was first described in the United States in the middle 1930's,[1,2] there has been a wealth of information published relative to its etiology, pathogenesis, radiographic studies, clinical signs, genetics, and medical management. Very little has been published on surgical treatments in the canine.

In the past there have been basically three surgical procedures used for treating dogs with hip dysplasia.[3,4,5,6] They are (1) acetabuloplasty, (2) pelvic osteotomy, and (3) excision of the femoral head and neck. The first two are limited to young dogs wherein advanced femoral head and acetabular changes have not occurred. The third is a salvage procedure.

In 1967, the pectineus tenactomy at the proximal end of the pectineus tendon of insertion and pectineus tenotomy at the pectineus tendon of origin were developed for treating clinically dysplastic dogs. These surgical procedures have been reported.[7,8,9] Since the pectineus tendon surgery was first developed, over 300 dogs have been operated on at the Kansas State University Small Animal Hospital.

B. Summary of Clinical Cases (Pre-Surgery)

1. Over 20 different breeds of dogs have been operated on.

2. Sex: About equal distribution of male and female.

3. Age at the time of surgery: 4 months to 12 years.

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4. Weight at the time of surgery: 30 to 210 pounds.

5. General health: All dogs have been in good to fair health. Those
   in fair health had cardiac, nutritional, neurologic, or other
   orthopedic problems at the time of surgery.

6. Duration of pre-surgical lameness: During the first 3 1/2 years,
   only dogs with a clinical lameness were operated on; and that ranged
   from two weeks to eight years. In the past year, several dogs have
   been operated on that according to the owners were not showing any
   clinical signs. These will be discussed later. At the other
   extreme are the dogs that were so disabled that they needed assis-
   tance to walk into the hospital; some had to be carried.

7. Progressive lameness: There may be several remissions and
   exacerbations of lameness in a dysplastic dog. Generally, after
   a period of time the lameness becomes more severe than it was on
   previous occasions.

8. Age when the first radiographic diagnosis of hip dysplasia was
   made: In our patients this has ranged from 4 months to 8 1/2 years.
   All grades of hip dysplasia have been operated on.

9. Joint laxity in clinically affected dogs: Joint laxity is checked
   for at the time radiographs are taken or prior to surgery while the
   patient is anesthatized. In younger dogs, varying degrees of coxo-
   femoral joint laxity are present. In older dogs with advanced
   osteoarthritic changes, joint laxity may not be detectable. This
   is basically due to a thickened joint capsule thus limiting lateral
   displacement of the femoral head from the acetabulum.

10. Disposition or mental attitude: Several dogs that have been
    operated on had developed severe personality changes. Most of

these dogs had at one time been even-tempered and playful and then gradually or suddenly became mean, frequently biting children and/or adults. These dogs also became less active and would not obey commands from the owners. Following surgery, the temperament of these dogs improved dramatically, and they once again became friendly, playful pets.

C. Radiology

Progressive radiographic changes can be expected after operating on dogs that are 5 months of age or older with minimal radiographic changes at the time of surgery. Although these progressive changes occur, the dogs have been rehabilitated and are clinically free of pain. (Figures 1-3) (Slides to be shown)

D. Gross Anatomy of the Pectineus Muscle

The pectineus muscle originates ventral to the acetabulum from a short thick tendon which has attachments to the prepubic tendon and adjacent ileo-pectineal eminence of the pelvis. The tendon of insertion lies between the adductor muscle caudally and the vastus medialis cranially. It inserts on the popliteal surface of the femur. The tendon of insertion is long, thin, and wide.

E. Surgical Procedure (Tendonectomy at the Proximal End of the Tendon of Insertion) (Figure 4) (Slides to be shown)

1. Bilaterally, the medial thigh and inguinal regions are prepared in a routine manner for aseptic surgery.

2. The dog is positioned in dorsal recumbency. The rear legs are secured in position with each femur perpendicular to the ventral midline. Positioning this way allows the pectineus muscle and tendon of insertion to be easily palpated. The caudal belly of the sartorius muscle will also have less tension on it and allow

it to be manipulated as described below. In most dogs the proximal circumflex femoral artery and vein can be observed crossing the distal end of the pectineus muscle. The caudal belly of the sartorius muscle can also be palpated as it obliquely crosses the distal end of the pectineus muscle from an anterior to posterior distal direction just distal to the above-mentioned blood vessels.

3. Skin drapes are placed anterior and posterior to each pectineus muscle and distally over each supracondylar region and secured with towel clamps. Draping in this manner facilitates going from one leg to another. A shroud can then be put over the surgical site.

4. For this procedure, the skin incision is started where the proximal circumflex vessels cross the pectineus muscle. It is continued distally for two inches staying over the pectineus tendon of insertion.

5. The subcutaneous tissue is bluntly dissected with scissors.

6. The caudal belly of the sartorius muscle is identified where it crosses the distal end of the pectineus muscle. Just proximal to where the sartorius muscle crosses the distal belly of the pectineus muscle, the proximal circumflex femoral vessels can be clearly identified.

7. From the point where the above-mentioned vessels are located, there is a dense fascia attached to the posterior edge of the caudal belly of the sartorius muscle. This fascia must be carefully incised, from where the sartorius crosses the pectineus, for one inch distad. Occasionally a small blood vessel is present in the above fascia going to that part of the sartorius muscle. That

blood vessel should be ligated. Be careful, as the main femoral artery and vein lie immediately under that part of the sartorius muscle. After the above-described fascial incision has been made, tissue forceps are applied to the fascia at the posterior edge of the sartorius muscle; and the sartorius is reflected anterior.

8. The femoral artery and vein are easily identified. Bluntly dissect the fascia along the posterior edge of the femoral vein and reflect it and the artery cranially along with the sartorius muscle.

9. The distal end of the pectineus muscle and the proximal end of its tendon of insertion will be clearly identified. Very carefully bluntly dissect the fascia between the pectineus and adductor muscle caudally and the vastus medialis anterior. Special care should be taken not to penetrate the fascia covering the adductor and vastus medialis muscles.

10. Retract the adductor muscle posteriorly. After clearly identifying the proximal end of the tendon of insertion and distal muscle belly of the pectineus, a curved Kelley forcep is passed anterior to posterior around and under the proximal end of the tendon of insertion. The tendon is then brought up to the level of the skin incision. The Kelley forceps are now effectively holding the femoral vessels and sartorius muscle out of the way.

11. The jaws of the Kelley forceps are opened wide. Tissue forceps are applied to the pectineus tendon of insertion between the open jaws of the Kelley forcep, about 1 cm. apart.

12. A tendonectomy is completed by severing the tissue between ths two tissue forceps. Make the first cut along the distal edge of the proximal forcep. The muscle will retract proximad. Roll the

distal forcep over in a distad direction and remove the remaining section of tissue. Palpate and visually inspect the surgical area to make sure all of the tendon was severed and excised. Digitally push the pectineus muscle further proximad in the leg.

13. It must be emphasized that all of the tendon must be severed for the surgery to be successful.

14. Because of the close proximity of the femoral vessels, do not try to close the dead space left from the tendonectomy.

15. Subcutaneous tissue and skin closures are rout'ne.

F. Surgical Procedure (Tenotomy of Pectineus Tendon of Origin) (Slides to be shown)

1. I prefer the tendonectomy that has been described above; however, this procedure at the tendon of origin has also been used with equally good results in dogs with advanced hip dysplasia.

2. Prepare and position the dog as described above.

3. A skin incision two inches long is made over the proximal end of the pectineus muscle. The subcutaneous tissue around the proximal end of the muscle is bluntly dissected.

4. The proximal end of the pectineus muscle and its tendon of origin are identified and isolated.

5. The deep femoral artery and vein are identified.

6. A curved Kelley forcep is passed around and under the extreme proximal end of the pectineus muscle in an anterior to posterior direction. The forceps should be between the deep femoral vessels and the muscle. Special care must be taken not to incorporate the deep femoral vessels in the forceps. Because of the anatomy of the area, it is impossible to get these forceps completely under the tendon of origin.

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7. A tenotomy is done through the tendon of origin.

8. Special care must be taken not to sever any of the prepubic tendon or the aponeurotic attachments of the abdominal muscles. To do this would facilitate the development of a hernia. It is therefore recommended that the tenotomy be done at the teno-muscular junction of the pectineus muscle.

9. Following tenotomy, the pectineus muscle will retract distad in the leg. It should also be digitally pushed further distad. It is recommended that the proximal end of the muscle be sutured to the subcutaneous tissue in the location to which it has been pushed. After doing the tenotomy, the area must be carefully inspected to make sure no fibers were left intact; otherwise, the surgery will not be successful.

10. Subcutaneous tissue and skin closures are routine.

G. Post-operative Care

1. Restrict exercise to a leash, and do not allow the dog to run free for 10 days.

2. Skin sutures can be removed on the tenth post-operative day after which there are no exercise restrictions.

3. Occasionally a seroma may develop at the surgical site of one or both legs. These are generally first noted by the owner between the third and twelfth post-operative days. If they do not become too large, no treatment is necessary as they will usually absorb in 14 to 21 days. If they become large and bother the dog, they can be aseptically aspirated. Seromas have been seen more frequently in the larger breeds of dogs.

H. Comment

This surgical procedure is not a cure for hip dysplasia. It is intended to relieve pain and rehabilitate the clinically dysplastic dog regardless of whether it is a family pet or a working dog. To date it has proven to be a highly effective surgical treatment in the dog with hip dysplasia too far advanced for either the acetabuloplasty or pelvic osteotomy procedures. In 4 1/2 years, I have only had to do one femoral head and neck resection in a dog that had the pectineus surgery. That was because of pathologic fractures in one femoral head that developed one month after the pectineus surgery. A very thorough physical examination of each patient prior to surgery is essential to make sure that a concomitant orthopedic or neurologic condition (fractures, joint injuries, spondylitis, metabolic or infectious bone disease, neuropathy, etc.) is not also present. If these other conditions are present, they will have to be treated. The owner must be informed that this operation is not intended to make the dog have a normal gait. Occasionally a younger dog, 5-6 months of age, may develop a normal or near normal gait; however, this cannot be predicted. Most of the dogs already have architectural changes in the coxofemoral articulation that biomechanically make it impossible for them to gait normally. The relief of pain is believed due to the release of tension on the pectineus muscle and the periarticular soft tissue of the coxofemoral articulation. It is impossible to know for how long a time this surgical procedure will hold up in a given dog. Over the past 4 1/2 years, it has been completely successful in alleviating pain and rehabilitating 94% of the dogs operated on. In all cases the owners are informed that the dog should not be used for breeding purposes. At the owner's request, orchiectomy, vasectomy, or ovariohysterectomy

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may be done at the same time as the pectineus tendon surgery. Several

questions have been raised regarding the feasibility of doing a myotomy

or myectomy of the pectineus muscle instead of the tendon surgery. A

pectineus myectomy can be done on a clinically dysplastic dog and

achieve the same results. However, the tissue space left is greater,

and the chances of seroma development are increased. A myotomy can also

be done as it allows tension release on the muscle and coxofemoral

periarticular soft tissue. The myotomy poses some inherent dangers.

'lealing together of the severed muscle ends is more likely to occur and

cause recurrence of pain and clinical lameness a few weeks or months

following surgery. I prefer and recommend the tendon surgery instead

of a myotomy.

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END

Illustration omitted

Sequential radiographs of the development of secondary arthritic changes of the acetabula and femoral heads following a bilateral pectineus tendonectomy for hip dysplasia. At the time of surgery the dog was $5\frac{1}{2}$ months old and very lame (Figure 1).

Illustration omitted

Early secondary arthritic changes of the anterior rim of the acetabula and
capital epiphyses of the femurs 4 months post-surgery (Figure 2).

Illustration omitted

Advanced secondary arthritic changes of the coxofemoral joints 34 months

post-surgery (Figure 3). The dog has been clinically sound since the

pectineus tendonectomy and has hunted vigorously each hunting season.

Illustration omitted

Figure 4--Drawing illustrating the tendon of origin (A), proximal end of
the tendon of insertion (B), and distal end of tendon of insertion (C) of
the pectineus muscle in the dog.